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STUDY OF ABG ANALYSIS IN RELATION TO SEVERITY OF ORGANOPHOSPHORUS POISONING

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ABSTRACT
Organophosphates exert their toxicity by interfering with acetylcholine by inhibiting acetyl cholinesterase's enzyme and many number of enzymes belonging to the group of carboryl esterases, paraoxonases (A-esterases) and other nonspecific proteases.Normally the enzyme acetyl cholinesterase hydrolyses the acetyl choline to two inactive fragments, choline and acetic acid, preventing continued stimulation of local receptors and eventual paralysis of the nerve and muscle. A detailed clinical examination of patients was done. In patients with respiratory failure, oxygen saturation using pulse oximeter, and with arterial blood gas analysis was done. Other relevant investigations available in the infrastructure was done wherever required. Grading of severity of poisoning was done according to clinical features and peradynia organ phosphorus scale, and those who require ventilatory support and those who did not require ventilatory support were assessed. Those who required mechanical ventilatory support, were put on mechanical ventilator and data accumulated was analysed with respect to above objectives. Out of 500 cases of ABG analysis, 71 cases showed respiratory acidosis of which 32 survived & 39 expired.210 cases showed respiratory alkalosis, of which 185 patients survived & 25 expired Irrespective of level of consciousness, clinical respiratory inadequacy, if ABG shows respiratory acidosis patients needs ventilatory support.

INTRODUCTION

Organophosphorus compounds are normally esters, amides or thiol derivatives of phosphoric or phosphonic acids or phosphorothinic or phosphonothocic acids. Most are only slightly soluble in water [1,2]. Organophosphates exert their toxicity by interfering with acetylcholine by inhibiting acetyl cholinesterase's enzyme and many numbers of enzymes belonging to the group of carboryl esterases, paraoxonases (A-esterases) and other nonspecific proteases. Normally the enzyme acetyl cholinesterase hydrolyses the acetyl choline to two inactive fragments, choline and acetic acid, preventing continued stimulation of local receptors and eventual paralysis of the nerve and muscle [3,4]. True cholinesterases are present in human nervous system, skeletal muscle, erythrocyte membrane and hence its activity can be assessed by RBC cholinesterase measurement, RBC cholinesterase activity levels parallels functioning nervous system acetylcholinesterases, whereas pseudocholinesterases in a hepatic derived protein that is found in human plasma, liver, heart, pancrease and brain. Its activity can be measured by plasma pseudocholinesterases level, although its endogeneons function is not fully understood. Organophosphorus bind irreversibly to cholinesterase, thus inactivating the enzyme through the process of phosphorylation, a process that normally takes 24-48 hours. This process is called "Ageing" and this

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period known as the critical interval because during this time administration of antidote is still effective in reversing the process. Once the ageing occurs however the enzyme activity of cholinesterase is permanently destroyed and new enzyme must be resynthesized over a period of weeks. Plasma Acetylcholinesterase recovers quickly usually within 4 weeks. Red cell Acetylcholinesterase takes longer and may not be restored to normal function for several months. Affected Acetylcholinesterase recovers at the rate of 1% per day. Ageing has an important bearing on toxicity and treatment outcome. Delayed neuropathic action of organophosphorous compounds is due to phosphorylation of neuropathy target esterases [5,6].

METHODOLOGY

Adult patients admitted with a history of consumption of organophosphorus compound poisoning during study period with characteristic signs and symptoms are included. Name of the compound confirmed by the empty bottles brought by the patient's relatives.

Exclusion Criteria

1. All patients with poisoning due to other than organophosphorus compound will be excluded.
2. Patients with poisoning due to mixed poison will be excluded.
3. Patients with history of chronic lung disease, COPD, asthma, bronchiectasis, extensive pulmonary tuberculosis and interstitial lung disease.
4. Patients with history of chronic cardiac disease.
5. Patients with history of chronic cardiac disease.
6. Patients with a history of neuromuscular disease.

A detailed clinical examination of patients was done. In patients with respiratory failure, oxygen saturation using pulse oximeter, and with arterial blood gas analysis was done. Other relevant investigations available in the infrastructure was done wherever required. Grading of severity of poisoning was done according to clinical features and peradynia organophosphorus scale, and those who require ventilatory support and those who did not require ventilatory support were assessed. Those who required mechanical ventilatory support, were put on mechanical ventilator and data accumulated was analysed with respect to above objectives.

Diagnosis

Diagnosis is based on

1. History of exposure to organophosphorus compound poisoning.
2. Characteristic manifestations of organophosphorus poisoning including miosis, fasciculations and excessive salvation.
3. Inhibition of cholinesterase activity.
4. Improvement after atropine and oxime therapy.
5. Corroborative evidence like empty containers with smell of organophosphorus compound.

All the cases satisfying inclusion and exclusion criteria were included in this study. A detailed pretested proforma was used to include relevant details of each patient such as name, age, sex, occupation, address, nature of poison and symptoms, and treatment interval and hospitalization treatment received before admission.

Depending on the severity of manifestations patients were classified into three grades as mild, moderate and severe (peradynia organophosphorus poisoning scale). Patients relevant past history, family history and personal history were also noted.

Investigations

Soon after admission in all patients included in this study, oxygen saturation using pulse oximeter and with arterial blood gas analysis were done. Patients who showed signs of respiratory failure were put on mechanical ventilation and monitored until patient recovered or till death.

Other Investigations

All routine investigations like complete blood count, blood urea, serum creatinine and serum electrolytes, ECG, chest X-ray whenever required. These tests were carried out in all patients.

All patients were managed with decontamination procedures including gastric lavage. IV atropine 2-4 mg bolus and repeated every 5-15 minutes until atropinization, sings of atropinization taken as the clear chest on auscultation, pupils-dilated and drying up of secretions. The atropinisation was maintained for 24-48 hours. Then tapered over days depending upon patients response.

Prolidoxime chloride was given to organophosphorus poisoning patients as 30 mg/kg loading dose bolus over 10-15 minutes immediately after admission and followed by a continuous infusion of 8-10 mg/kg per hour until clinical recovery or 7 days whichever is later.

Patients airway and need for mechanical ventilatory support were assessed and in needed cases mechanical ventilatory support were given. Follow up of cases was done regarding response to treatment and follow up of cases on mechanical ventilatory support, until discharge or death of the patient. The duration of hospital stay and in hospital outcome was documented.

RESULTS

Table 1. Place distribution

Place		Male		Female	Total	
	No.	%	No.	%	No.	%
Urban	27	6.8	13	13	40	8
Rural	373	93.2	87	87	460	92
Total	400	100	100	100	500	100

Majority of the patients were from rural areas. It may be due to their occupation agriculture and easy availability of organophosphorus compound to rural people.

Table 2. Treatment interval and its relation to severity of poisoning

Treatment		Mild		Moderate		Severe		Total
(hours)	No.	%	No.	%	No.	%	No.	%
< 2	30	13.7	4	1.9	0	0	34	6.8
2 to 4	173	79	126	60	9	12.7	308	61.6
4 to 6	15	6.8	55	26.2	33	46.5	103	20.6
> 6	1	0.5	25	11.9	29	40.8	55	11
Total	219	100	210	100	71	100	500	100

p-value = 0.000, Highly significant

As the treatment interval increases, severity of poisoning also increases. This was statistical significant.

Table 3. Treatment interval and its relation to outcome

Treatment		Expired	Improved			Total
(hours)	No.	%	No.	%	No.	%
< 2	0	0	34	7.8	34	6.8
2 to 4	8	12.5	300	68.8	308	61.6
4 to 6	23	35.9	71	16.3	94	18.8
> 6	33	51.6	31	7.1	64	12.8
Total	64	100	436	100	500	100

p-value = 0.000, Highly significant

The majority of patients were hospitalised within 2-4 hours of exposure and in this group the mortality (12.5%) was seen as compared to mortality (35.9%) among patients who were hospitalised between 4 and 6 hours after exposure and mortality in patients who were admitted after more than 6 hours it was 51.6%. This difference was statistically significant. As the treatment interval increases poor will be the outcome.

Table 4. Relation between oxygen saturation and the development of respiratory failure

Oxygen saturation	Total number of patients	Number of patients developed	Percentage
< 90%	145	145	100
> 90%	355	0	0
Total	500	145	100

If the oxygen saturation is < 90% the proportion of patients developing RF also increases.

Table 5. Severity of grading of poisoning and its relation to outcome

Grading	Improved			Expired		Total
	No.	%	No.	%	No.	%
Normal	219	50.2	0	0	219	43.8
Respiratory alkalosis	185	42.4	25	39.1	210	42.0
Respiratory acidosis	32	7.4	39	60.9	71	14.2
Total	436	100	64	100	500	100

Out of 500 cases of ABG analysis, 71 cases showed respiratory acidosis of which 32 survived & 39 expired.210 cases showed respiratory alkalosis, of which 185 patients survived & 25 expired.

DISCUSSION

Most of the patients in the present study were belonged to the low socio-economic status, farmers, housewives and labourers. Similar findings were reported by Goel et al [7] and Gannur DG et al [8]

Table 6. Place distribution

Place	Present study	Dash SK et al
Urban	8%	20%
Rural	92%	80%

Majority of the patients (92%) in this study were from rural areas. This is comparable to studies by Dash SK et al [9]

Dimethoate is the commonest organophosphorus used in this study, whereas methyl parathion in a study by Palimar et al.[10] This may be due to regional differences.

Respiratory failure was the most common complications seen in 29% of cases (including respiratory failure as a consequence of intermediate syndrome). This is comparable to studies by Sungur et al.[11] and Cherian MA et al.[2]
In the present study, intermediate syndrome was noticed in 12.8% of patients is comparable to studies by Sungur et al.[11] and Senanayake et al.[12]

Table 7. Outcome of ventilatory support

Total number	Expired	% of mortality	Sungur et al.[11]		Palimar et al.[10]
Respiratory failure	81	48	59%	50%	-
Intermediate syndrome	64	16	25%	-	22.3%

Mortality rate in the present study was 59% in respiratory failure and 25% in intermediate syndrome after mechanical ventilation. This is comparable to Sungur et al.[11] and Palimar et al.[10]

Out of 500 cases of ABG analysis, 71 cases showed respiratory acidosis of which 32 survived & 39 expired.210 cases showed respiratory alkalosis, of which 185 patients survived & 25 expired So respiratory acidosis has high mortality.

CONCLUSION

Ventilatory support cannot be guided by clinical assessment of respiratory inadequacy but guided only by ABG analysis. In our study conscious patient with adequate respiratory effort showed respiratory acidosis by ABG analysis. So these patients also needed ventilator support.

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CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

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